Ocular manifestations of mercury poisoning

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One hundred patients suffering from organomercury poisoning who were hospitalized in the Medical City, University of Baghdad, were examined ophthalmologically in the period between March and December 1972, and were reviewed again about 10 months later.

The examination included clinical signs and symptoms, slit lamp examination of the anterior segment of the eyes, examination for visual field changes and, in the autopsied cases, analysis of the optic nerve and the occipital cortex. In addition, aqueous humour was aspirated in 10 patients and in 10 controls for the estimation of the mercury level.

RESULTS

The onset of ocular signs and symptoms was usually early, although these were not the earliest signs of poisoning. Only occasional patients stated that blurring of vision was the first symptom and in these cases it occurred at the same time as numbness, ataxia, and other neurogenic symptoms.

A few patients mentioned xanthopia as the beginning of the visual disturbance. Two patients had experienced recurrent attacks of visual loss that eventually culminated in persistent visual loss.

Most patients gave a history of 1-4 weeks of exposure and of the onset of symptoms 10-30 days after exposure ceased.

The severity of the visual loss varied, even between the patients from families who seemed to have been exposed to the same dose. Often a patient was completely blind but had a brother or sister who shared the same home but who had no detectable visual disturbance.

There was no very close correlation between the degree of visual disturbance and the level of blood mercury, as estimated in the early weeks of hospitalization, but most of the patients who suffered visual disturbance had a blood mercury level above 100 ng/ml at the time of onset of symptoms. No visual disturbance could be detected in patients who had a blood mercury level of less than 500 ng/ml early in March 1972, 2-3 weeks after the onset of symptoms.

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Patients with severe visual loss did not necessarily have higher levels of blood mercury than patients with lesser visual defects. It seems that the duration of exposure and probably the amount of ingested methylmercury may influence the severity of the visual disturbance (Table 1).

TABLE 1. VISUAL LOSS AND CONCENTRATIONS OF MERCURY IN BLOOD AS ESTIMATED IN THE FIRST MONTH AFTER ADMISSION

Blood mercury (ng/ml)	Patients											
	Exposure period (days)	Blind		Severe visual loss		Moderate visual loss		Slight visual loss		No visual loss		Total No.
		No.	(%)[a]	No.	(%)[a]	No.	(%)[a]	No.	(%)[a]	No.	(%)[a]	
10-500	41	-	-	-	-	-	-	-	-	11	(100)	11
501-1 000	45	3	(16)	1	(6)	2	(11)	7	(39)	5	(28)	18
1 001-2 000	45	9	(35)	5	(19)	5	(19)	3	(11)	4	(16)	26
2 001-3 000	55	7	(35)	5	(25)	1	(5)	7	(35)	-	-	20
3 001-4 000	58	9	(60)	4	(27)	2	(13)	-	-	-	-	15
4 001-5 000	68	7		3	(30)	-	-	-	-	-	-	10
Total No. of patients		35		18		10		17		20		100

[a] % of total in blood mercury group.

In this study 34 patients were found to be severely affected, totally blind or with only perception of light. A further 18 patients had very poor vision; these could count fingers only at less than 6 m. Sixteen patients had moderately affected vision, while 12 had slight disturbance such as 6/9 6/9 visual acuity or 6/12 in the worse eye. In 20% of the patients there was no visual loss. Half of these complained only of slight numbness of hands, while the other half suffered moderate ataxia and slurred speech.

Visual field defects were more striking than impairment of visual acuity, the most common finding being concentric contraction of the fields. The central vision was almost always spared. A few patients showed hemianopia. In over one-third of the cases the visual fields could not be assessed, either because of the gross impairment of vision or because of the apathetic condition of the patient.

Thirty cases showed gross contraction of visual fields. The optical area was central, round, with steep borders. These patients were the most visually disabled, although several of them had 6/9 or 6/12 central visual acuity. Moderate visual field defects were present in 25 cases.

Corneal changes did not seem to be related to mercury, apart from the loss of corneal sensation in the worst affected patients. Blind patients with less severe systemic and neurologic signs did not show absent or blunted corneal sensations. Similarly, 95 cases had no abnormalities in the lens.

The anterior chamber appeared clear and healthy in all patients. No flare could be detected even in the seriously affected cases. The aqueous humour was aspirated in 10 cases with a variable degree of visual loss. The results are shown in Table 2.

TABLE 2. MERCURY LEVEL OF AQUEOUS HUMOUR (AH) IN 10 PATIENTS: RELATIONSHIP TO BLOOD MERCURY LEVEL AND DEGREE OF DISABILITY

AH Hg ng/ml	Blood Hg ng/ml	Ratio AH:Blood	Visual defect
11	1 453	0.007	Blind
11	1 374	0.008	Blind
30	142	0.21	Slight CVF[a]
42	634	0.06	Slight CVF
47	439	0.11	Severe CVF
63	595	0.11	Moderate CVF
65	525	0.12	Severe CVF
73	1 990	0.04	Severe CVF
91	1 440	0.06	Severe CVF
104	878	0.12	Slight CVF

[a] CVF = constriction of visual fields.

The ciliary body is not a barrier to the mercury. All the specimens from affected patients showed a high mercury level, but this was always lower than that of the blood. There was no relationship with the severity of visual defects.

A semidilated pupil with a sluggish or absent reaction to light was seen in children totally blind or with perception of light only. Adults had normally functioning pupils. Two children were found to have patches of iris atrophy and one case had juxtapapillary choroiditis. We believe these to be coincidental findings.

One patient had complete paralysis of accommodation, with no other abnormal findings in the eyes. Most of the patients complained of difficulties in picking out small objects, but evaluation of accommodation was difficult because most of the victims were illiterate.

On fundoscopy, most of the patients showed slate pigmentation around the optic disk, but in most cases changes in the disk itself were minor or absent. Ten patients with severely affected vision had a hyperemic disk with an ill-defined margin and with swelling ranging between 1-1/2 and 2 diopters.

In a number of blind children, pallor of the optic disk was seen. Several cases showed sheathing of the juxtapapillary blood vessels.

Most of the blind and visually disabled adults showed narrowing of the retinal blood vessels and rather waxy looking disks with a diminished number of arterioles but no change in colour.

Marked optic atrophy was observed in some of the otherwise moderately affected children under 12 years of age. We could not detect any optic atrophy in some of the teenagers with severe visual defects and saw only one case in a blind young adult.

The retina appeared pale in cases of severe visual loss both in children and adults, but the macular area was usually healthy. Despite the various motor and sensory disturbances that were present in the patients with mercury poisoning, extraocular movements were

always normal. The severely affected patients who were completely paralysed and apathetic showed random movements of the eyes and most of them had blepharospasm when ocular examination was attempted.

Despite the gross ataxia of some patients, no nystagmus was noticed in any of the cases, even in young children with very poor vision or in babies born blind to affected mothers.

On follow-up some patients showed slight improvement and enlargement of the visual fields that had been constricted. A few improved from perceiving only light to just seeing hand movement or counting fingers if these were placed close to the eye. Three children could move about much better than a few months before, and no longer bumped into objects. However, the majority of the patients remain blind or seriously disabled. None of the seriously affected patients improved to the point of normal vision. The condition of one patient with monocular vision who initially had depression of the visual field and visual acuity of 6/12, appeared to have worsened to 6/18 with further restriction of the visual fields when he was re-examined 3 months later.

DISCUSSION

The visual field changes in most of the mercury-poisoned patients examined appeared to be constriction of all quadrants to within a certain degree of the fixation point. In those patients who showed gross constriction no improvement was found on follow-up examination. However, some patients, totally blind initially, could perceive light or see movement of hands when re-examined after a few months. There were no marked changes in fundoscopy, except for diminution of the retinal blood vessels.

The pattern of the restriction of the visual fields resembles that of quinine and arsenical amblyopia, except that in the latter an oval-shaped field is more usual. In quinine amblyopia marked optic atrophy is seen, but here cases of optic atrophy were infrequent and were confined to young children.

At one stage it was considered that loss of vision in severely affected cases might be attributed to central pathological change and in most of the moderately affected cases might be due to lesions in the optic nerve fibres. Pallor of the retina and the partial optic atrophy, as well as the narrowing of blood vessels and sheathing of the bases of the vascular tree, suggested a peripheral cause of blindness. However, when autopsy was carried out on a baby who was blind before death, the optic nerve contained more mercury than the temporal lobe and about the same level as that in the occipital lobe. Autopsy of a blind adult revealed much less mercury in the optic nerve than in the occipital lobe. Therefore, this hypothesis could not be maintained.

RESUME

MANIFESTATIONS OCULAIRES DE L'INTOXICATION PAR LES COMPOSES MERCURIELS

Cent malades souffrant d'intoxication par organomercuriels, hospitalisés à la Cité médicale de l'Université de Bagdad, ont subi des examens ophtalmologiques de mars à décembre 1972 et des examens de contrôle une dizaine de mois plus tard.

On a observé les signes et symptômes cliniques, examiné le segment antérieur de l'oeil à la lampe à fente, contrôlé les modifications du champ visuel et, chez les sujets autopsiés, analysé le nerf optique et le cortex occipital. En outre, on a procédé à l'aspiration d'humeur aqueuse chez 10 malades et 10 témoins pour estimation de la concentration de mercure.